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Bacteremia Caused by *Achromobacter* Species in an Immunocompromised Host

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A case of bacteremia caused by *Achromobacter* species in an immunocompromised patient is described. The patient responded to antibiotic therapy. Detailed antibiotic susceptibility data are presented.

Unusual organisms are often opportunistic pathogens in immunocompromised patients; in particular, nonfermenting, gram-negative rods are being reported with increasing frequency. One such group of organisms belongs to the genus *Achromobacter*. Members of this genus possess peritrichous flagella, do not ferment glucose, and are oxidase positive. The genus *Achromobacter* consists of one species, *Achromobacter xylosoxidans*, and unnamed strains presently designated *Achromobacter* spp. or CDC group Vd (6). *A. xylosoxidans* has been isolated as a pathogen from numerous sources (4–6). However, except for one case report of a pancreatic abscess caused by *Achromobacter* spp. biovar 1 (1), little information is available concerning strains designated as *Achromobacter* spp. We describe a case of bacteremia caused by *Achromobacter* spp. and present detailed antimicrobial susceptibility data for this isolate.

Case summary. A 16-year-old woman developed a seizure disorder in 1977 and was discovered to have a grade II astrocytoma of the right frontal lobe. This was resected with incomplete margins, and the patient received postoperative radiation therapy. The patient did well until September 1981 when a right temporal lobe recurrence was diagnosed. Over the next 6 months, the patient received four courses of intracarotid chemotherapy (right-sided) with *bis*-chloroethylnitrosourea. This treatment regimen resulted in steroid-responsive cerebral edema, chronic pancytopenia, and no appreciable reduction in tumor bulk.

In June 1982, the patient developed fever, chills, and left flank pain and was admitted to the hospital. The medications on admission included (milligrams per day): dexamethasone (4), diphenylhydantoin (400), carbamazepine (1,200), and valproic acid (750). Temperature was 103.5°F (ca. 39.7°C), pulse was 72/min, and blood pressure was 100/60 mm Hg. No skin rash or adenopathy was present. An infraclavicular Hickman catheter site (inserted 1 month earlier for vascular access) was without erythema, tenderness, or purulence. Lungs were clear; a grade 2/6 systolic ejection murmur was present and unchanged from previous exams. Abdominal and pelvic exams were normal. The patient had mild, left costo-vertebral angle tenderness. Mental status was normal. Neurological exam revealed mild, left hemiparesis with bilaterally upgoing toes. Urinalysis revealed no erythrocytes or bacteria and five to seven leukocytes per high-power field. Chest X ray was normal. Serum creatinine was 0.9 mg/dl. Liver function tests were normal. Hemoglobin was 9.5 mg/dl; hematocrit was 28% in volume. Platelet count was 90,000/mm^3, and leukocyte count was 1,800/mm^3, with 50% neutrophils, 21% band forms, 36% lymphocytes, and 13% monocytes.

Upon admission of the patient, therapy was initiated with 18 g of ticarcillin and 210 mg of tobramycin per day. The patient quickly became afebrile, and the flank pain disappeared. Urine culture was negative. However, three sets of blood cultures drawn at the time of admission grew a gram-negative, oxidase-positive, nonfermenting rod within 24 h. This oxidase-positive organism, which grew on MacConkey agar, was classified in the genus *Achromobacter* on the basis of its ability to reduce nitrate and oxidize xylose. In addition, the isolate was indole and lysine decarboxylase negative and was unable to oxidize lactose or sucrose. The organism was subsequently identified as *Achromobacter* spp. (group Vd), rather than *A. xylosoxidans*, on the basis of a positive urea reaction within 18 h and the detection, by using a lead acetate paper strip, of H_2S production. Because the organism was resistant to ticarcillin and tobramycin, these antibiotics were discontinued. Gentamicin at 270 mg/day and trimethoprim-sulfamethoxazole at eight ampoules per day (80 mg of trimethoprim and 400 mg of sulfamethoxazole per ampoule) were given intravenously for the next 2 weeks. The patient remained afebrile, and blood cultures drawn during and at the completion of antibiotic therapy were negative.

Antimicrobial susceptibility data. The MIC and MBC were determined for 32 antibiotics by using a microtiter broth dilution technique (3). The MICs and MBCs of penicillin G, ampicillin, methicillin, nafcillin, dicloxacillin, ticarcillin, carbenicillin, piperacillin, mezlocillin, cephalothin, cefazolin, cefamandole, cefoxitin, cefotaxime, ceftriaxone, cefoperazone, vancomycin, clindamycin, netilmicin, and trimethoprim were >100 μg/ml. The MICs and MBCs of the remaining antibiotics tested are shown in Table 1.

Discussion. Some information regarding the antimicrobial susceptibility of *Achromobacter* spp. in a disk diffusion test has been reported (2). By this method of susceptibility testing, the organism appeared susceptible to gentamicin, amikacin, tetracycline, trimethoprim-sulfamethoxazole, and colistin. To our knowledge, there are no data from an MIC-MBC method of testing susceptibility. From our MIC data, it appeared that gentamicin, trimethoprim-sulfamethoxazole, and tetracycline were again the most active agents in vitro.

It should be noted, however, that the MBC exceeds the clinically achievable level in serum for all three of these

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TABLE 1. Antibiotic susceptibilities of *Achromobacter* spp.

Antibiotic[a]	MIC (μg/ml)	MBC (μg/ml)
Erythromycin	25	>100
Moxalactam	12.5	50
Streptomycin	25	100
Gentamicin	1.6	12.5
Tobramycin	25	50
Amikacin	25	>100
Imipenem	3.1	25
Tetracycline	<0.2	50
Chloramphenicol	50	>100
Polymyxin B	0.4	100
Rifampin	12.5	25
Trimethoprim-sulfamethoxazole	<2-<38	>128->2,432

[a] Twenty other antibotics were tested and had MICs and MBCs of >100 μg/ml (see text).

antibiotics. Moxalactam and imipenem also showed activity against this isolate, and the MBC for these antibiotics can be achieved in serum. Our patient appeared to respond to ticarcillin and tobramycin, to which the organism from the patient had exhibited resistance in vitro. Before the MBC data was known, the therapy of the patient was changed to gentamicin and trimethoprim-sulfamethoxazole, and the patient did well. In vitro testing does not always correlate with the in vivo response; however, it would have been more appropriate to use antibiotics the MBCs of which were in the clinically achievable range. In an immunocompromised patient, host factors cannot be relied upon to help control the infection, and it may be more important to use antibiotics which have bactericidal activity against the infecting organism.

Achromobacter spp. have been isolated from a number of clinical sources, including wounds, urine, sputum, and blood (2, 6), but information with respect to its pathogenicity in humans is lacking. The natural habitat of the organism has not yet been established, but aqueous environments have been implicated (1, 6). The recovery of *Achromobacter* species from this patient was an isolated event and not part of a hospital-wide outbreak. It is possible that the patient had either a urinary tract infection with subsequent seeding of the bloodstream or a spontaneous bacteremia from her own enteric flora. The chronic neutropenia and steroid therapy may have been predisposing factors which allowed successful infection with this organism.

With continuing advances in cancer chemotherapy and organ transplantation and the resulting immunosuppression in these patients, clinicians will continue to see infections caused by unusual organisms. The reporting of data on clinical syndromes caused by such organisms and antibiotic susceptibilities of such organisms will assist clinicians and microbiologists in the care of patients with opportunistic infections.

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